PLAINTIFF'S REPLY EXHIBIT 1

**UNITED STATES DISTRICT COURT
SOUTHERN DISTRICT OF WEST VIRGINIA
AT CHARLESTON**

**WEST VIRGINIA RIVERS
COALITION, INC.,**

Plaintiff,

v. **Civil Action No. 2:24-cv-00701**

THE CHEMOURS COMPANY FC, LLC,

Defendant.

DECLARATION OF JENNIFER SCHLEZINGER, PH.D.

I, Jennifer Schlezinger, do hereby certify, swear or affirm, based on my personal knowledge unless otherwise stated, that the following statements are true and correct to the best of my knowledge:

1. I am a resident of East Falmouth, Massachusetts.
2. I am over the age of 18, and I am competent to make this Declaration.
3. I make this Declaration in support of the West Virginia Rivers Coalition's Reply in Support of its Motion for a Preliminary Injunction.
4. I am a Professor of Environmental Health at Boston University School of Public Health with more than 25 years of experience as a research scientist in public health.
5. I have a BA in Biology from Boston College. I have a PhD in Biological Oceanography from the Massachusetts Institute of Technology and Woods Hole Oceanographic Institution Joint Program. I was trained in toxicology while in my doctoral program and completed a research dissertation on the toxicology of polychlorinated biphenyls.

6. My research has focused on the molecular toxicology of contaminants found in the indoor and outdoor environment. I have published more than 70 manuscripts in this field.

7. I am an internationally recognized expert in the toxicology of per- and polyfluoroalkyl substances ("PFAS"), as evidenced by publication of my studies in international journals and invitations to present my research at the annual meetings of the Society of Toxicology (an international professional society). I study how PFAS, including hexafluoropropylene oxide dimer acid ("HFPO-DA"), cause adverse health effects. I also develop and use models that are designed to maximize the generation of human-relevant data on PFAS toxicity.

8. I have significant experience in assessing the human relevance of toxicological data as evidenced by my participation in the International Agency for Research on Cancer's reassessment of the human carcinogenicity of perfluorooctanoic acid and perfluorooctane sulfonic acid and recent publication on the cardiovascular disease risk factors associated with PFAS exposure.

9. My curriculum vitae, a copy of which is attached, outlines my professional experience, including education, training and research experience along with my publications, which positions me to assess the merit of the toxicological questions raised in this case.

10. In connection with this matter, I have reviewed the following:

   - Defendant's March 11, 2025 Response in Opposition to Plaintiff's Motion for a Preliminary Injunction. Civil Action No. 2:24-cv-00701 (ECF No. 17).

   - The March 11, 2025 Declaration of Catherine Boston in Support of Chemours' Response in Opposition to Plaintiff's Motion for a Preliminary Injunction (and the Exhibits thereto) (ECF No. 17-7).

- Human Health Toxicity Values for Hexafluoropropylene Oxide (HFPO) Dimer Acid and Its Ammonium Salt (CASRN 13252-13-6 and CASRN 62037-80-3). EPA Document Number: 822R-21-010, dated October 2021.
- Drinking Water Health Advisory: Hexafluoropropylene Oxide (HFPO) Dimer Acid (CASRN 13252-13-6) and HFPO Dimer Acid Ammonium Salt (CASRN 62037-80-3), Also Known as "GenX Chemicals." EPA Document Number: EPA/822/R-22/005, dated June 2022.
- PFAS National Primary Drinking Water Regulation, 89 FR 32532-01
- Analytic Search Results | SDWARS. Available at https://lubeckpsd.com/Portals/LubeckPSD/UCMR5_Results.pdf
- All of the scientific literature cited elsewhere in this declaration and in the References.

11. Based on my review of this information and the scientific literature, and my education, knowledge, research and experience, it is my professional opinion that the contamination of the Lubeck Public Service District's drinking water with HFPO-DA related to The Chemours Company FC, LLC ("Chemours") discharges of wastewater from the Washington Works Plan in Washington, West Virginia, to the Ohio Rivers increases risk of adverse human health effects.

12. I base my professional opinion on the following:

13. Ms. Boston categorically states (in paragraphs 12 and 29 excerpted below) that there is no potential for adverse human health effects resulting from the contamination of the Lubeck Public Service District's drinking water supply with HFPO-DA released from the Washington Works Plant in Washington, West Virginia.

¶ 12. "[T]here are no potential adverse human health effects resulting from alleged contamination of hexafluoropropylene oxide dimer acid (HFPO-DA) in the Lubeck Public Service District's drinking water related to the Chemours Company, LLC ("Chemours") discharges of wastewater from the Washington Works Plant in Washington, West Virginia to the Ohio River."

¶ 29. "Thus, there is no scientific support for claims of potential adverse human health effects resulting from alleged contamination of HFPO-DA in the Lubeck Public Service District's drinking water related to Chemours's discharges of wastewater from the facility."

These statements are wrong and unsupported by the science.

14. First, there is ample scientific evidence that HFPO-DA is toxic and growing evidence that it is more toxic than perfluorooctanoic acid ("PFOA"), the chemical that also has contaminated the Lubeck Public Service District's drinking water from releases from the Washington Works Plant in Washington, West Virginia. The EPA identified 75 studies from which it was concluded that HFPO-DA induces adverse effects in the liver, hematological system, and immune system and is a developmental toxicant.[1] Since March 2020, an additional 17 studies on the *in vivo* toxicity of HFPO-DA have been published (identified by searching Pubmed: hfpo-da AND 2020/03/01:2025/3/15[dp] AND toxic*). Here I highlight important new findings. Studies continue to support that disruption of liver function is a sensitive endpoint of HFPO-DA toxicity in rodent models.[2–9] Studies in human liver cell models show that HFPO-DA causes changes in the genes that are expressed,[10,11] which corroborate the findings in rodent studies. Additionally, studies show that toxic changes in cells and organs may be more readily

initiated by HFPO-DA than by PFOA or perfluorooctane sulfonic acid (the PFAS currently considered to be the most toxic).[2,12]

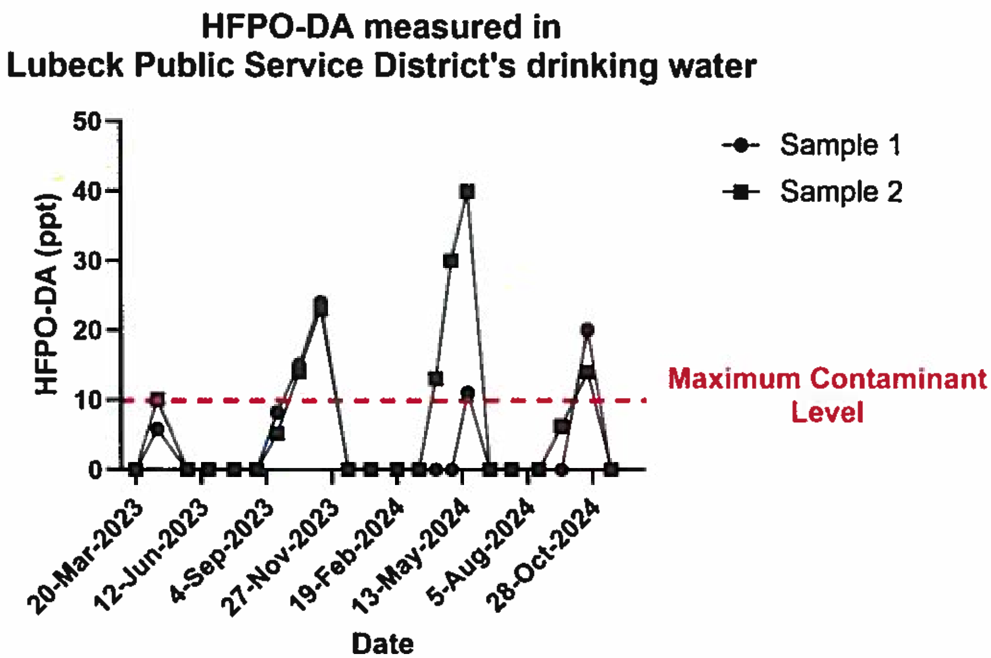
15. Chemours (in ¶ 8 of their response) noted that "Crucially, HFPO-DA levels in Plaintiff's member's water supply are under EPA's Maximum Contaminant Level." In line with this, Ms. Boston stated (at ¶ 26), "Although there are some individual sampling results in the Lubeck public drinking water system that are elevated above 10 ng/L, compliance with the MCL value of 10 ng/L is dependent on a long-term annual average, and short-term spikes in effluent values are compensated for by the vast majority of the sampling results below 10 ng/L, or ND for HFPO-DA. This includes the period in April 2024, where any short-term elevation in HFPO-DA levels would not have been of a sufficient duration or magnitude to result in a risk to human health."

The statement that "any short-term elevation in HFPO-DA levels would not have been of a sufficient duration or magnitude to result in a risk to human health" is wrong and unsupported by the science.

16. Although the HFPO-DA concentrations may be compliant with the annual averaging requirements of EPA's maximum contaminant level ("MCL"), that is not determinative of the question of the existence of risk. Note that when Ms. Boston calculated the average HFPO-DA concentrations to analyze if they were below the MCL, she did so by averaging all the values in a given year, rather than using the quarterly value average approach used by the EPA to determine compliance with the regulation.[13]

17. Using the monthly monitoring data from samples taken after the "lag" bed and provided in Table 18 of Attachment B to Ms. Boston's declaration, reflected in paragraph 20 of

that declaration, I generated the following plot demonstrating the significant and sustained contamination events that occurred over 2023-2024.



The maximum contaminant level is a drinking water concentration designed as a maximum safe concentration level based on a reference dose (the maximum amount of a toxicant that can be consumed daily over a lifetime without an increase in risk of adverse health effect). Stated otherwise, a person needs to drink water with less than 10 ppt of HFPO-DA every day for a lifetime for there to be no increase in risk of adverse health effect. It is clear from the data that this reference dose-derived safe level has been exceeded multiple times and over long periods. It is a false equivalent to say that no harm has occurred because the yearly average exposure remained below the MCL. For instance, a 14-week exposure to a low level of HFPO-DA in drinking water in a rodent study showed that the development and function of the placenta was disrupted.[14] Furthermore, HFPO-DA was found to accumulate over time in amniotic fluid and in

fetuses over time;[15] and the developing human fetus is at its greatest susceptibility to toxic effects in a 5 week period of time between 3-8 weeks of gestation.[16] Thus, extended periods of exposure are not required for HFPO-DA to increase the risk for adverse human health effects.

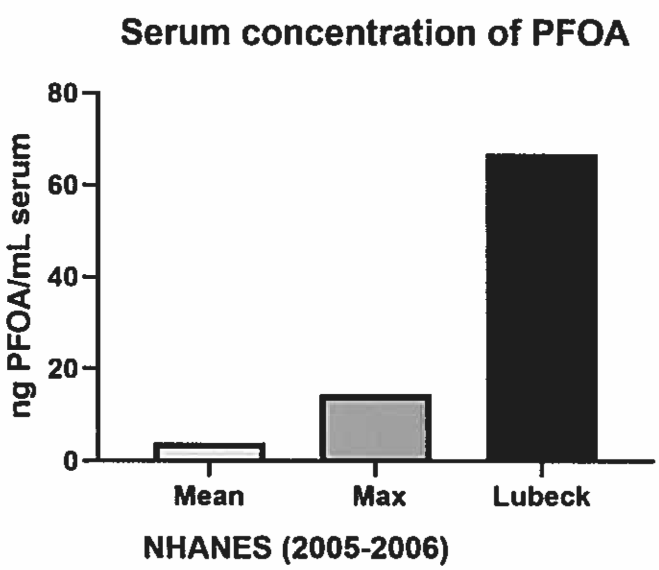
18. Ms. Boston states (¶ 28), "[T]he MCL was derived using incredibly conservative assumptions to ensure protection of public health."

This statement is also wrong and unsupported by the science.

19. The procedure followed by the EPA in defining the reference dose for HFPO-DA, which is the basis for the MCL, was standard practice.[1,17] Adverse endpoints in the liver were chosen for the dose response assessment that defined the point of departure. Liver effects are observed in both female and male rodents at multiple durations of exposures and doses. For HFPO-DA, the benchmark dose was used, which addresses the major shortcoming of the reference dose approach. Further, the point of departure calculation took into account the human equivalent dose. Four standard uncertainty factors were applied: 10 for interhuman variability, 3 for interspecies variability, 10 for extrapolation from a sub-chronic to chronic exposure duration, and 10 to account for database deficiencies. An uncertainty factor for database deficiencies was applied because the EPA could not rule out the possibility that developmental/placental endpoints could be more sensitive to toxic effects of HFPO-DA than liver. Developmental deficits and placenta lesions have been observed following HFPO-DA exposure, but 2-year developmental/reproductive studies have yet to be conducted and endpoints likely shared with PFOA (e.g., incomplete bone formation) have yet to be analyzed. The dose response analysis and application of uncertainty factors used the most recent data and followed standard procedures, and as such, is completely reasonable and not "incredibly

conservative." Indeed, Baird et al (1996) found that application of a total uncertainty value of 3000, with the application of 4 uncertainty factors, provides approximately 99% confidence of adequate—not excess—conservatism.[18]

20. Ms. Boston's opinion that there is no potential for adverse human health effects resulting from exposure to HFPO-DA over the MCL at the Lubeck Public Service District's drinking water supply is also wrong because it ignores that the community served by the Lubeck Public Service District's drinking water has already been excessively exposed to PFOA. Below is a plot of the serum concentration of perfluorooctanoic acid in the general US population (as determined in the National Health and Nutrition Examination survey)[19] and in Lubek residents[20] following the contamination of the water supply by the Washington Works Plant, in Washington, WV. The people of Lubeck had PFOA levels in their blood that were 17x higher than the average level in the American population.



In the absence of further contamination of the water supply with PFOA, it could be expected that the PFOA body burden of people living in Lubeck would have eventually decreased. However, data show that exposure to PFOA continues in this community (see

¶ 23 of Ms. Boston's response). The Lubeck Public Service District reports that concentrations of PFOA in domestic water it supplied to its customers reached at least 179.5 ppt during a valve failure of a treatment system.[21] Thus, the people of Lubeck are carrying a higher-than-average body burden of a toxic PFAS. This is relevant for three reasons:

- Exposures to mixtures of PFAS can cause combined toxic effects.
- The RfD for HFPO-DA was not determined within a model or population with an existing PFOA exposure.
- The United States National Academies of Science, Engineering and Medicine recognized the need to consider multiple PFAS exposures when developing guidance for clinicians.

21. *Exposure to mixtures of PFAS causes combined effects.* In a cell model system, activation of human PPARα was shown to be activated by a mixture of PFAS in an additive/combined manner.[22] Further, human liver spheroids exposed to PFAS mixtures were shown to change the expression of genes in an additive/combined manner.[23] Gray and Conley (2021) also indicated that interactions of PFAS (including PFOA and HFPO-DA) produce combined toxicity that is greater than the toxicity of each individual PFAS.[24]

22. *The RfD for HFPO-DA was not determined with a model or population with an existing PFOA exposure*. As described above (¶ 18), the reference dose was determined from a study carried out by Dupont in which neither PFOA nor HFPO-DA levels in blood of the rodents was reported. The potential for co-exposure to PFOA to impact the assessment of a safe dose of HFPO-DA has not been applied. The EPA does consider noncancer health

risks from PFAS mixtures and uses the health index approach for estimating the risk of exposure to multiple PFAS including combined exposures to PFOA and HFPO-DA.[25]

23. *The United States National Academies of Science, Engineering and Medicine (NASEM) recognized the need to consider multiple PFAS exposures when developing guidance for clinicians.* NASEM recently provided clinical guidance for follow up with patients exposed to the seven PFAS most commonly measured in Americans.[26] The consensus report recommends that individuals with summed serum concentrations of NASEM PFAS ≥ 20 ng/ml should undergo clinical follow-up for thyroid function (age 18 or older); kidney function and cancer (age 45 or older); and ulcerative colitis and testicular cancer (age 15 or older). Individuals with the sum of the same PFAS ≥ 2 ng/ml should be prioritized for screening for dyslipidemia, hypertensive disorders of pregnancy, and breast cancer (at specified intervals). While HFPO-DA was not included in the current clinical guidance, the importance of considering combined exposures to PFAS when considering the risk adverse health effects is clear.

24. Chemours asserts that because the annual average HFPO-DA levels were below the USEPA MCL in the Lubeck Public Service District's drinking water, that there is no scientific support for claims of potential adverse human health effects resulting from alleged contamination of HFPO-DA in the Lubeck Public Service District's drinking water related to Chemours's discharges of wastewater from the facility. As outlined above, this is a flawed analysis of the circumstances as it ignores the fact that MCLs are concentrations based on references doses, which define maximum levels of safe daily consumption. In its Final MCL Rule, EPA expressly noted that the "annual average" element of compliance measurement includes a "buffer" that incorporates concepts of "operational certainty" and "operational flexibility" and that "could allow some results to

exceed [the MCL] for single measurements if the overall annual average is below the MCL." 82 Fed. Reg. at 32574. In other words, it appears that the "annual average" component was based in part on policy considerations, rather than scientific ones. Because the HFPO-DA concentrations in the Lubeck Public Service District's drinking water exceed the safe concentration repeatedly, the people of Lubeck are experiencing increased risk of adverse health effects resulting from Chemours discharges of wastewater from the Washington Works Plant in Washington, West Virginia.

All of my opinions in this matter are within a reasonable degree of scientific certainty. I declare under penalty of perjury that the foregoing is true and correct.

Executed on March 18, 2025

Jennifer Schlezinger, PhD

Responses in Livers of Mice Exposed to the Short-Chain PFAS Compound HFPO-DA. *Front Toxicol.* 2022;4:937168. doi:10.3389/ftox.2022.937168

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9. Attema B, Janssen AWF, Rijkers D, van Schothorst EM, Hooiveld GJEJ, Kersten S. Exposure to low-dose perfluorooctanoic acid promotes hepatic steatosis and disrupts the hepatic transcriptome in mice. *Mol Metab.* 2022;66:101602. doi:10.1016/j.molmet.2022.101602

10. Heintz MM, Klaren WD, East AW, et al. Comparison of transcriptomic profiles between HFPO-DA and prototypical PPARα, PPARγ, and cytotoxic agents in mouse, rat, and pooled human hepatocytes. *Toxicol Sci.* 2024;200(1):165-182. doi:10.1093/toxsci/kfae044

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24. Gray E, Conley J. Characterization of developmental toxicity and Adverse Outcome Pathways for emerging PFAS - individual compounds and mixtures. In: *SOT Risk Assessment and Mixtures Specialty Sections Joint Webinar Series.* ; 2021. https://cfpub.epa.gov/si/si_public_record_Report.cfm?Lab=CPHEA&dirEntryID=351857

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Curriculum Vitae
Jennifer J. Schlezinger, Ph.D.
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Department of Environmental Health
715 Albany Street, R408
Boston, MA 02118

ACADEMIC TRAINING:

1998 Ph.D.	Biological Oceanography, Woods Hole Oceanographic Institution/Massachusetts Institute of Technology Joint Program, Woods Hole, MA
1992 B.S.	Biology, Boston College, Chestnut Hill, MA

POSTDOCTORAL TRAINING:

2000-2003	Postdoctoral Fellow, Department of Environmental Health, Boston University School of Public Health, Boston, MA
1998-2000	Postdoctoral Trainee, Immunology Training Program, Boston University School of Medicine, Boston, MA

AREAS OF SPECIALIZATION:

Molecular toxicology (nuclear receptors; liver, bone and adipose toxicology; organotins; PFAS)

ACADEMIC APPOINTMENTS/FACULTY MEMBERSHIPS:

2024-present	Professor of Environmental Health, Boston University School of Public Health, Boston, MA
2009-present	Faculty Member, Molecular Medicine, Division of Graduate Medical Sciences, Boston University School of Medicine, Boston MA
2010-2024	Associate Professor of Environmental Health, Boston University School of Public Health, Boston, MA
2012-2015	Director of Master of Science Program in Environmental Health, Boston University School of Public Health, Boston, MA
2008-2016	Faculty Member, Immunology Training Program, Boston University School of Medicine, Boston, MA
2005-2010	Assistant Professor of Environmental Health, Boston University School of Public Health, Boston, MA

2003-2005	Research Assistant Professor of Environmental Health, Boston University School of Public Health, Boston, MA

DEPARTMENTAL AND UNIVERSITY COMMITTEES:

2024-present	Faculty Senate, Department representative
2021-present	Academic Conduct Committee member, Boston University School of Public Health
2012-present	Laboratory Safety Committee member, Boston University
2012-present	Doctoral/MSc/Post-Doctoral Committee member, Department of Environmental Health, Boston University School of Public Health
2021-2024	Laboratory Safety Committee Chairperson, Boston University
2020-2022	Search Committee member, Opportunistic Hiring Committee for Department of Environmental Health, Boston University School of Public Health
2020-2021	Laboratory Safety Committee Vice Chairperson, Boston University
2017-2021	LSC Subcommittee Chairperson (High Hazard Chemicals/Chemical Safety)
2018-2020	Faculty Senate, At large member
2012-2019	MSc Committee member, Boston University School of Public Health
2012	Teaching Award Selection Committee member, Boston University School of Public Health
2011-2015	Core Advisory Committee member, Boston University Medical Campus
2010	Search Committee member, Director of Faculty Development, Boston University School of Public Health
2008	Search Committee member, Associate Dean for Research, Boston University School of Public Health
2007-2010	Faculty Senate Vice Chairperson, Boston University School of Public Health
2006-2012	Curriculum Committee member, Department of Environmental Health, Boston University School of Public Health
2006-2008	Institutional Animal Care and Use Committee member, Boston University Medical Campus

TEACHING EXPERIENCE:

2023-present	Faculty Co-leader for *Module 1 – Data, Determinants and Decision-Making for Health Equity* (SPH OM701)
2023-present	Director for *Toxicology for Environmental Health and Epidemiology* (SPH EH768)
2019-present	Instructor for *Individual, Community and Population Health* (SPH PH720)
	2020: Excellence in Teaching Award for Teaching in the Core
2017-present	Director for *Toxicology for Public Health* (SPH EH705)
2022-2023	Guest lecturer for *Essentials of Public Health* (SAR PH510)

2010-2019	Director for *Advanced and Emerging Topics in Toxicology* (SPH EH840)
	2011: Excellence in Teaching Award
2017-2018	Director for *Essentials of Toxicology* (SPH, online short course)
2013-2016	Director for *Introduction to Toxicology* (SPH EH768)
2007-2016	Lecturer in *Comprehensive Immunology* (GMS MI713)
2007-2011	Co-director for *Molecular Biology in Public Health* (SPH EH713)
	2011: Excellence in Teaching Award
2006-2008	Instructor in *Intermediate Toxicology* (SPH EH840)
2002-2005	Lecturer in *Molecular Biology in Public Health* (SPH EH713)

STUDENT MENTORING:

2020-present	Ph.D. Co-Mentor (Microbiology, Scott Adams)
2011-present	Ph.D. qualifying exam committee member (Environmental Health: Kevin Lane; Program in Biomedical Sciences: Essence Maston; Program in Biomedical Sciences: Kathrine Benson; Program in Biomedical Sciences: Thomas Shin; Program in Biomedical Sciences: Grant Duclos; Program in Biomedical Sciences: Jiarui Zhang; Bioinformatics: Eric Reed; Program in Biomedical Sciences: Jordan Shafran; Biology: Shalom Entner; Environmental Health: Kathryn Rogers)
2009-present	Master of Public Health advisor (Environmental Health: Susan Yanik, Rachael Brem, Elizabeth Faye, Alejandra Ramirez-Cardenas, Lauren Buck, Aliya Zwyer, Keyana White, Tanmayee Karanam, Raina Levin, Lindsay Kastner, Peyton Comfort)
2008-present	Practicum supervisor (Environmental Health: Elizabeth Clarke, Hari Pillai, Lauren Masse, Heather McKenney, Rachel Freid, Catherine Boston, Hannah Puckett, Lauren Payne, Audrey Garcia)
2006-present	Doctoral committee member. Boston University - Environmental Health: Gregory Howard; Pathology: Supraja Narasimhan; Ashley Penvose: Computational Biomedicine: Eric Reed; Biology: Shalom Entner. UMASS Lowell – Pharmaceutical Sciences: Kushal Biswas. University of Louisville – Pharmacology and Toxicology: Morgan Delnicki.
2019-2023	Ph.D. Mentor (Environmental Health, Greylin Nielsen). Scientist: MA Department of Environmental Protection
2015-2019	Ph.D. Mentor (Environmental Health, Stephanie Kim)
	Karen Wetterhahn Memorial Award recipient, Post-doc: USEPA
2014-2019	Ph.D. Co-Mentor (Environmental Health, Lariah Edwards), Employment: Gradient Consulting Post-doc: George Washington University/Environmental Defense Fund
	EPA STAR Fellowship recipient
2013	Master of Science research mentor (University of Paris, Diderot, Lydie Barbeau)
2014-2018	Ph.D. Co-Mentor (Environmental Health, Kate Crawford), Post-doc: Dartmouth University
2012-2016	Ph.D. Mentor (Environmental Health: James Watt), Post-Doc: Louisianna State University

2012-2016	Second reader for Ph.D. thesis (Molecular Medicine: Ashley Parks, Olga Novikov)
2010-2019	Master of Science advisor (Environmental Health Data Analytics: Paige Brochu, Jennifer Rooney; Payton de la Cruz; Environmental Health: James Watt, Charlotte Marsh, Natalie Banacos; Graduate Medical Sciences: Holly Bernard)
2010-2013	Ph.D. committee chairperson (Molecular Medicine: Brian Sworder, Anna Belkina)
2009-2011	Directed Research advisor (Environmental Health: Susan Yanik, James Watt)
2008-2012	Ph.D. mentor (Molecular Medicine: Amelia Haas)
	Individual NRSA Fellowship recipient
2008	Outside reader for Ph.D. thesis (Experimental Medicine: Zuanel Diaz Heredia, McGill University)
1999-2009	Second reader for Master of Medical Sciences theses (Todd Jenkins, Pamela Bernard, Olga Novikov)

PROFESSIONAL MENTORSHIP

2020-present	External advisory committee member (Abby Fleisch, Tufts University, ONES)
2023-present	Mentor (Jamie Young, University of Louisville, K01)
2021	Mentor (Jamie Young, University of Louisville, DP5)

OTHER PROFESSIONAL SERVICE:

MEMBERSHIPS

2022-present	American Heart Association
2012-present	Boston Nutrition Obesity Research Center
1998-present	Society of Toxicology
2010-2015	American Society for Bone and Mineral Research

OFFICES/POSITIONS

2024-Present	Society of Toxicology, Vice-President, Mixtures Specialty Section
2023-Present	Society of Toxicology, Board of Publications
	Member – 2023-2025
	Co-Chair – 2025-2026
	Chair – 2026-2027
2023-2024	Society of Toxicology, Vice-President Elect, Mixtures Specialty Section
2021-2023	Society of Toxicology, Secretary/Treasurer, Mixtures Specialty Section
2017-2019	Society of Toxicology, Counselor, Mixtures Specialty Section
2016-2018	Society of Toxicology, Counselor, Metals Specialty Section

Advisory Boards

2025-Present	Toxics Use Reduction Act (TURA) Program, Science advisory board member

Peer Review Activities

2022-present	*Environmental Health Perspectives*, Editorial Review Board, Member
2000-present	Manuscript reviewer: Aquatic Toxicology, Chemosphere, Environmental Health, Environmental Health Perspectives, Environmental Science and Technology, Environmental Toxicology and Applied Pharmacology, Journal of Immunology, Journal of Orthopedic Research, Journal of Toxicology and Environmental Health, Molecular Pharmacology, Scientific Reports, Toxicological Sciences, Toxicology
2025 (March)	NIH *ad hoc* Peer Reviewer: Environmental Determinants of Disease
2024 (July)	NIH *ad hoc* Peer Reviewer: Special Emphasis Panel: Member Conflict: Topics in Hepatology, Pharmacology, and Toxicology
2024 (March)	NIH *ad hoc* Peer Reviewer: Special Emphasis Panel: Member Conflict: Topics in Hepatology, Pharmacology, and Toxicology
2022, 2023	NIH *ad hoc* Peer Reviewer: Cell Signaling and Molecular Endocrinology
2022	NIH *ad hoc* Peer Reviewer: Special Emphasis Panel: Systemic Injury by Environmental Exposure
2021	NIH *ad hoc* Peer Reviewer: Special Emphasis Panel: Functional Genomics for Interrogating GxE Interactions in Disease
2021	Intramural Site Visit Review of NCI's Laboratory of Metabolism
2019, 2021	Superfund Research Program Review Panel
2019	Netherlands Organisation for Scientific Research, Reviewer for Veni grant in the Innovational Research Incentives Scheme, entitled: 'FOCUS: Finding the Origins of Chemical-Induced Obesity'
2019	ViCTER Review Panel
2015-2019	NIH/NIEHS - Environmental Health Sciences Review Committee
2018	Adverse Outcome Pathway reviewer for the Organisation for Economic Co-operation and Development (OECD). Reviewed the AOP for Antagonist of PPARα.
2016, 2018	NIH/NIEHS – K99/K00 (Pathway to Independence) Review Committee
2014	NIH Peer Review Committee *ad hoc*: The Role of Environmental Exposures in the Development of Autoimmune Disease (ES-13-011)

Meeting/Symposium Chairships

2024 Society of Toxicology Annual Meeting Symposium: *Mechanisms of Per and Polyfluorinated (PFAS) Action…PPARα and Beyond*

Co-Chairperson

2024 Society of Toxicology Annual Meeting Symposium: *The "Cocktail Effect": Studying the Greatest Uncontrolled Experiment Ever Launched!*

Co-Chairperson

2019 Society of Toxicology Annual Meeting Poster Session: *Receptors*

Co-Chairperson

2013 Society of Toxicology Annual Meeting Symposium: *Bone as a Target Tissue for Environmental Toxicants*

Co-Chairperson

2012 Northeast Superfund Research Translation Activity: *Complex Mixtures and Exposures: Analyzing, Modeling and Predicting Fate and Effects at Multiple Levels of Environmental and Biological Systems*

Co-Chairperson

RESEARCH SUPPORT:

CURRENT SUPPORT

2025-2029 R01 HL173978

NIH/HLBI

Identifying Environmental Pollutants Detrimental to the Cardiovascular System

Role: Co-Principal Investigator (Gokce – Co-PI)

The goal of this project is to use a combined clinical and basic/molecular investigation to examine mechanisms of vascular dysfunction associated with persistent organic pollutants and environmental toxicants that contribute to the pathogenesis of cardiovascular diseases.

Total Award Amount: $2,797,661

2024-2026 R21 ES035475

NIH/NIEHS

Optimizing a Human-Relevant Mouse Model to Study Adverse Health Effects of PFAS

Role: Principal Investigator

The goal of this project is to further refine the hPPARα mouse model I developed to examine PFAS-induced health effects by crossing it with the hPXR/hCAR mouse.

Total Award Amount: $453,750

2023-2026 23CSA1052192

American Heart Association

	Adverse vascular effects of environmental pollutants
	Role: Co-Principal Investigator (Gokce – Co-PI)
	The goal of this project is to characterize, to our knowledge for the first time, direct pathogenic effects of diverse toxicants, both legacy (banned) and presently widely used, upon the human vascular system by probing direct and indirect mechanisms owing to disruption of adipocyte biology.
	Total Award Amount: $858,444
2023-2026	HT94252310690
	DOD/TERP
	A Novel and Practical Intervention for Detoxification of PFAS in Humans
	Role: Principal Investigator
	The goal of this project is to test the hypothesis that consumption of gel-forming and positively charged natural fibers will reduce PFAS absorption and increase PFAS elimination. And, as a result, PFAS-induced adverse health effects also will be minimized.
	Total Award Amount: $1,117,352
2023-2025	R21 ES034939 (Gokce, PI)
	NIH/NIEHS
	Impact of Per/Polyfluoroalkyl Pollutants on Vascular Disease Mechanisms
	Role: Co-Investigator
	The goal of this proposal is to define the pathophysiological effects of structurally diverse PFAS upon the human vascular system
	Total Award Amount: $453,750
2022-2024	R03 ES034093
	NIH/NIEHS
	Investigating the Perturbation of Bone Health by Per/Polyfluoroalkyl Substances
	Role: Principal Investigator
	The goal of this project is to define the effect of long term PFOA exposure on cortical and trabecular structure, osteoblast and osteoclast number and function, and the bone transcriptome.
	Total Award Amount: $165,000

SUPPORT PENDING

SUPPORT UNDER REVIEW

2025-2027	NIH/NIEHS
	Untangling the Immunotoxicity of PFAS Exposure on Susceptibility to Respiratory Infection

Role: Co-Principal Investigator (Doaum – Co-PI)

The goal of this project is to test the hypotheses that: 1) PFAS exposure potentiates IFNγ and overall antiviral responses against SARS-CoV-2 infection but also increases susceptibility to re-infection through inducing lower humoral responses; and 2) PFAS-mediated protection is driven by enhanced IFNγ production and an American diet. Responses to SARS-CoV-2 will be compared to InfA.

Proposed Direct Costs: $250,000

2024-2027 DOD/TERP

Approaches to Reduce Cardiovascular Toxicity of Pyrethroid Insecticides in Military Personnel

Role: Principal Investigator

Use of insecticidal chemicals on uniforms is an essential strategy to reduce exposure of military personnel to insect-borne disease. The goal of this project is to test the hypotheses that exposure to military-relevant doses of permethrin promotes atherosclerosis. Furthermore, we hypothesize that the toxicity of permethrin is modulated by its formulation, dehydration and heat stress and that etofenprox may be a safer alternative.

Proposed Direct Costs: $800,000

PAST SUPPORT

2021-2024 R21 ES032882

NIH/NIEHS

Defining the Impact of Per/Polyfluoroalkyl Substance Exposure on Susceptibility to SARS-CoV-2 Infection and Disease

Role: Co-Principal Investigator (Doaum – Co-PI)

The goal of this project is to test the hypothesis that PFAS exposure enhances susceptibility to SARS-CoV-2 infection via interaction with nuclear receptors.

Total Award Amount: $444,650

2017-2023 R01 ES027813 (Webster, PI)

NIH/NIEHS

Novel Analytical and Experimental Approaches for Predicting the Biological Effects of Mixtures

Role: Co-Investigator

The goal of this project is to test the efficacy of generalized concentration addition as a model of additivity for complex mixture exposure scenarios.

2012, 2017 NIEHS/NTP

Targeted Testing of Adiogenic ToxPi Compounds

	Role: PI
	The goal of this project is to test the accuracy of the ToxCast approach and ToxPi method to predict the adipogenic capacity of toxicants.
2012-2017	P42 ES07381 (Ozonoff, PD)
	NIH/NIEHS Superfund Hazardous Substances Basic Research Program
	Environmental PPARγ Agonist-Mediated Toxicity in the Developing Immune System (Project 4)
	Role: Project Leader
	The goal of Project 4 is to test the hypothesis that environmental PPAR/RXR ligands suppress B lymphopoiesis by two mechanisms: directly, by inducing apoptosis in early B cells; and indirectly, by altering the bone marrow microenvironment that supports lymphopoiesis, resulting in aging-like suppression of immune responses.
2012-2014	R21 ES021136
	NIH/NIEHS
	Effects of High Fat Diet and Environmental Obesogen Co-Exposure on Osteoporosis
	Role: Principal Investigator
	The goal of this study is to examine how exposure to environmental obesogens (tributyltin) and a high fat diet cooperate to impair osteogenesis.
2011-2012	Ellison Foundation (Gerstenfeld, PI)
	Mechanism(s) of Obesity-Related Osteoporosis in the Absence of Insulin Resistance
	Role: Co-Investigator
	The goal of this study is to examine the connection between obesity and bone loss in a unique insulin-competent but obese mouse model, the BrD2 hypomorphic mouse.
2009-2011	R21 CA134882
	NIH/NCI
	Antagonism of the Ah Receptor in Controlling Breast Cancer Growth and Invasion
	Role: Principal Investigator
	The goal of this study is to screen natural products libraries for novel, high affinity AhR antagonists and to determine molecular mechanisms of AhR antagonist-mediated breast cancer growth suppression.
2005-2011	P42 ES007381 (Ozonoff, PD)
	NIH/NIEHS Superfund Basic Research Program at Boston University
	Environmental Phthalate- and PPARγ-Mediated Toxicity in the Developing Immune System (Project 4)
	Role: Project Leader

The goal of Project 4 is to map the apoptotic pathways induced by PPARγ agonists in the developing immune system and to investigate the how PPAR and RXR agonists synergize to induce apoptosis.

2004-2009 RO1 ES06086 (Sherr, PI)

NIH/NIEHS

How Environmental Chemicals Impair Immunity

Role: Researcher

The goal of this study is to investigate how polycyclic aromatic hydrocarbons agonists compromise immune function, with emphasis on mechanisms of induction of B cell apoptosis.

2000-2005 P42 ES007381 (Ozonoff, PD)

NIH/EPA Superfund Hazardous Substances Basic Research Program

The Ah Receptor as a Regulator of Hydrocarbon Bioactivity

Role: Researcher

The goal of this study was to assess the functional role of AhR in PAH responsiveness of mature lymphocytes and evaluate AhR polymorphism and expression in humans

2000-2001 Individual National Research Service Award

NIH/NIEHS

Aryl Hydrocarbon Receptor and NF-κB Interactions

Role: Post-doctoral fellow

The goal of this study was to investigate how AhR agonists alter the NF-κB signaling pathway in bone marrow stromal cells.

1998-2000 Institutional National Research Service Award

NIH/Oncobiology

Mechanisms of PAH-Induced, Bone Marrow Stromal Cell Mediated B Cell Death

Role: Post-doctoral trainee

The goal of this study was to examine the PAH-induced pathway leading to production of a B cell death signal by bone marrow stromal cells.

BIBLIOGRAPHY

INVITED PRESENTATIONS

1. The Multi-Faceted Toxicity of PFAS: Structures, Targets and Adverse Health Outcomes

University of Louiville. June 2024

University of Massachusetts, Lowell. September 2024.

University of Arizona. November 2024.

2. Is PPARα the MIE driving human relevant toxic effects of PFAS? Invited talk. *CPAC Joint Symposium on PFAS and Cancer.* Georgetown University. Washington, DC. March, 2024
3. What should we be thinking about (but aren't) in investigating PFAS-induced lipid disruption? Invited Seminar.

   Brown University, Providence, RI. February 2023.

   Wayne State University, Detroit, MI. March 2023.
4. PFOA induces liver and serum dyslipidemia in a humanized PPARα mouse model fed an American diet. *FLUOROS Conference*. Providence, RI. March 2021
5. PFOA induces liver and serum dyslipidemia in a humanized PPARα mouse model fed an American diet. Invited seminar. University of New Mexico School of Medicine, Albuquerque, NM. October 2021.
6. Environmental PPARγ and RXR ligands: selective agonists that disrupt bone and adipose homeostasis. Invited presentation. *Gordon Research Conference on Cellular and Molecular Mechanisms of Toxicity.* Andover, NH. August 2019.
7. Environmental PPARγ ligands: inducers of white, but not brite, adipogenesis. Invited seminar.

   Boston University, Biology Department, Boston, MA. October 2018

   Boston Nutrition Obesity Research Center, Boston, MA. December 2019.

   Baylor College of Medicine, Houston, TX. March 2020.

   Texas A&M University, College Station, TX. September 2020.
8. Environmental PPARγ ligands: inducers of white, but not brite, adipogenesis. Invited presentation *Society of Toxicology Annual Meeting*, San Antonio, TX. March 2018
9. Tributyltin suppresses B cell development by inducing apoptosis and altering the bone marrow microenvironment. Invited presentation. *American Association of Immunologists Annual Meeting*. Washington, D.C., 2017.
10. Approaches to studying multi-faceted exposures to nuclear receptor ligands. Invited presentation. *NIEHS FEST,* Durham, NC. December 2016.
11. Environmental PPARγ agonists: What are they doing to our bone and metabolic health? Invited seminars.

    McMaster University, Ontario, Canada. May 2015.

    McGill University, Quebec, Canada, May 2015.

    Boston Nutrition Obesity Research Center, Boston, MA. June 2015.

    Woods Hole Oceanographic Institution, Toxicology Roundtable, Woods Hole, MA. August 2015.

    Louisiana State University, Baton Rouge, LA. September 2016.
12. Environmental obesogens: are they bad to the bone? Invited seminar. University of Maine, Orono, MA. December, 2012.
13. Environmental contaminants skew mesenchymal stem cell differentiation in bone marrow: examining accelerated aging of bone. Invited seminar. Brown University, Providence, RI. May, 2010.
14. Environmental PPARγ agonists alter bone marrow mesenchymal stem cell differentiation and lymphopoiesis. Invited seminar. University of Massachusetts, Dartmouth, MA. March, 2010.

15. Environmental contaminants accelerate aging of bone: what may this mean for the immune system? Invited seminar. University of New England College of Medicine, Biddeford, ME. January, 2010.
16. Premature aging in bone: environmental chemical-induced effects on bone marrow adipogenesis and osteoblastogenesis. *Superfund Basic Research Program Annual Meeting*, New York, NY. November, 2009.
17. Environmental phthalate-mediated toxicity in developing B cells: interactions with endogenous PPARγ and RXRα agonists. *Superfund Basic Research Program Annual Meeting*, Monterey, CA. December, 2008.

PRESENTATIONS AND PUBLISHED ABSTRACTS

PLATFORM PRESENTATIONS

1. **Schlezinger, JJ.** 2024. Is PPARα the MIE driving human relevant toxic effects of PFAS? *Society of Toxicology Annual Meeting*, Salt Lake City, UT.
2. Nielsen, G, Reed, E, Webster, TF, Heiger-Bernays, W, Lara, B, Sherr, D, Hyötyläinen, T, **Schlezinger, JJ**. 2023. A multi-omics assessment of the effects of PFOA treatment on liver lipid homeostasis in a human relevant mouse model. *Society of Toxicology Annual Meeting*, Nashville, TN.
3. **Schlezinger, JJ**. 2022. PFOA induces liver and serum dyslipidemia in a humanized PPARα mouse model fed an American diet. *The Science of PFAS: Public Health and the Environment.* Meeting hosted by NEWMOA. Marlborough, MA.
4. **Schlezinger, JJ.** 2022. Sex-differences, an understudied factor in PFAS-induced liver toxicity and lipid disruption. *Society of Toxicology Annual Meeting*, San Diego, CA.
5. **Schlezinger, JJ**, Hyötyläinen T, Boston C, Heiger-Bernays W, Webster, TF. 2020. PFOA induces liver and serum dyslipidemia in a humanized PPARα mouse model fed an American diet. *Society of Toxicology Annual Meeting*, Anaheim, CA.
6. Webster TF, Hyötyläinen T, Boston C, Heiger-Bernays W, **Schlezinger J.** 2019. PFOA induces liver and serum dyslipidemia in a humanized PPARα mouse model fed an American diet. *Dioxin International Symposium, Kyoto, JP.*
7. **Schlezinger, JJ**, Watt, J, Gerstenfeld, LC. March 2015. Suppression of osteogenesis by organotins. Is it all about PPARγ? *Society of Toxicology Annual Meeting*, San Diego, CA.
8. Pillai H, Fang M, Beglov D, Kozakov D, Vajda S, Stapleton HM, Webster TF, **Schlezinger JJ**. June 2014. Firemaster 550 contains PPARγ ligands that induce adipogenesis and suppress osteogenesis. *14th Annual Workshop on Brominated & Other Flame Retardants*, Indianapolis, IN.
9. Watt, J, Andrews, F, **Schlezinger, JJ**. March 2013. Environmental obesogens: are they bad to the bone? *Environmental Health Conference,* Boston, MA.
10. **Schlezinger, J**, Baker AH, Gerstenfeld, LC. March, 2013. Organotins: Unique modifiers of bone quality and the bone marrow microenvironment. *Society of Toxicology Annual Meeting*, San Antonio, TX.
11. Webster, TF, Howard, GJ, **Schezinger, J**. March, 2008. Mixtures of AhR full agonists, partial agonists and antagonists: Implications for TEFs. *Society of Toxicology Annual Meeting*, Seattle, WA.
12. **Schezinger, J,** Bissonnette, S, Teague, JE, Sherr, DH. March, 2008. Contribution of p53 and BCL-2 family members in prostaglandin and phthalate-induced pro/pre-B cell apoptosis. *Society of Toxicology Annual Meeting*, Seattle, WA.
13. **Schlezinger, J,** Bissonnette, S, Sherr DH. March, 2007. GW7845, a PPAR agonist, induces mitochondria-dependent apoptosis in pro/pre-B cells. *Society of Toxicology Annual Meeting*, Charlotte, NC

14. **Schlezinger, JJ**, Emberley, J, Sherr, DH. March, 2005. PPARγ agonists, GW7845, 15-deoxy-$\delta^{12,14}$-Prostaglandin J_2, and mono-(2-ethylhexyl) phthalate, activate complex caspase cascades in pro/pre-B cells. *Society of Toxicology Annual Meeting*, New Orleans, LA.
15. Sherr, DH, Ryu, H-Y, Emberley, JE, Allan, LL, **Schlezinger, JJ**. March, 2005. AhR control of B lymphocyte death and growth. *Society of Toxicology Annual Meeting*, New Orleans, LA.

Poster Presentations

1. Ekuban, FA, Nielsen, G, Gondim, DD, Cave, MC, Heiger-Bernays, W, Webster, TF, **Schlezinger, JJ**. PFOA exposure results in sex-dependent effects on hepatic and whole-body lipid homeostasis that differ by PPARa status. *American Association for the Study of Liver Diseases Annual Meeting*, Boston, MA. 2023.
2. Doherty, A, **Schlezinger, J**, Kuohung, W. The effect of PFAS compounds on trophoblast migration and differentiation in placental cell lines. *Society for Reproductive Investigation Annual Meeting*, Denver, CO, 2022.
3. Nielsen, G, Heiger-Bernays, W, Webster, T, **Schlezinger, J**. Predicting the effect of PFAS mixtures on nuclear receptor activity. *Society of Toxicology Annual Meeting,* Virtual, 2021.
4. **Schlezinger, JJ**, Hyötyläinen, T, Sinioja, T, Puckett, H, Oliver, J, Heiger-Bernays, W, Webster, TF. PFOA modifies fatty acid and triglyceride homeostasis in a humanized PPARα mouse model fed an American diet. *Society of Toxicology Annual Meeting,* Virtual, 2021.
5. **Schlezinger, JJ**, Heiger-Bernays, W, Webster, TF Predicting the activation of the androgen receptor by complex mixtures of environmental antagonists using Generalized Concentration Addition. *Society of Toxicology Annual Meeting,* Virtual, 2021.
6. **Schlezinger, JJ**, Heiger-Bernays, W, Webster, TF. Modeling induction of proximal and distal endpoints following PPARγ activation by ligand mixtures. *Society of Toxicology Annual Meeting*, Anaheim, CA, 2020.

   Best Five Mixtures Poster winner
7. **Schlezinger, JJ**, Hyötyläinen, T, Boston, C, Heiger-Bernays, W, Webster, TF. PFOA induces liver and serum dyslipidemia in a humanized PPARα mouse model fed an American diet.

   SETAC North America Focused Topic Meeting: Environmental Risk Assessment of *PFAS*, Durham, NC, 2019.

   SETAC North America 40th Annual Meeting, Toronto, Ontario, Canada, 2019.
8. **Schlezinger, JJ**, Heiger-Bernays, W, Webster, TF. Predicting the activation of the androgen receptor by mixtures of ligands using Generalized Concentration Addition. *Society of Toxicology Annual Meeting*, Baltimore, MD, 2019.

   Best Mixtures Poster winner
9. Kim, S, Rabhi, N, Farmer, S, **Schlezinger, J**. Triphenyl phosphate, an environmental contaminant, is a selective PPARγ ligand that may not be so brite! *Society of Toxicology Annual Meeting*, Baltimore, MD, 2019.
10. Watt, J, Baker, AH, Freid, R, Hussein Ali, A, Divieti Pajevic, P, Morgan, EF, Gerstenfeld, LC, **Schlezinger, JJ**. Tributyltin increases trabecular bone in female C57BL/6J mice and protects against ovariectomy-induced trabecular bone loss. *American Society of Bone and Mineral Research Annual Meeting*. Montreal, Quebec, Canada, 2018.

11. Edwards, L, Webster, TF, **Schlezinger, J**, Peters, J. Investigating the association of a biomarker of triphenyl phosphate exposure with metabolic health in the U.S. population. *International Society for Environmental Epidemiology Annual Meeting*. Ottowa, Ontario, Canada, 2018.
12. Freid, R, Kim, S, Hussein Ali, A, **Schlezinger, J**. Tributyltin protects against ovariectomy-induced bone loss only in mice on a low fat diet. *Society of Toxicology Annual Meeting*, San Antonio, TX, 2018.
13. Edwards, L, Webster, T, Hatch, E, Janulewicz, P, **Schlezinger, J**. Assessment of mouse and human serum cumulative, ligand-induced peroxisome proliferator activated receptor γ agonist activity**.** *Society of Toxicology Annual Meeting*, San Antonio, TX, 2018.
14. Kim, S, Reed, E, Monti, S, **Schlezinger, J**. Application of Digital Gene Expression to identify adipogenic gene signatures of environmental metabolism-disrupting chemicals. *Superfund Research Program Annual Meeting*, Philadelphia, PA, 2017.
15. Edwards, L, Webster, T, Hatch, E, Janulewicz, P, **Schlezinger, J**. Assessment of mouse and human serum cumulative, ligand-induced peroxisome proliferator activated receptor γ agonist activity**.** *Superfund Research Program Annual Meeting*, Philadelphia, PA, 2017.
16. Crawford, K, **Schlezinger, J**, Nacci, D, Hahn, M, Clark, B, Heiger-Bernays, W. Healthy fish, healthy people: Using fish to understand ecological and human health impacts of early life exposures to Superfund chemicals on metabolic and bone development. *Society of Environmental Toxicology and Chemistry Annual Meeting.* Mineapolis, MN. 2017.
17. Schlezinger, J, Marsh, C, Kim, S, Heiger-Bernays, W. The organophosphate flame retardant triphenyl phosphate disrupts bone re/modeling in adult and perinatally exposed female mice. *Society of Toxicology Annual Meeting*, Baltimore, MD. 2017
18. Veltre, D, Chen, E, Hussein, A, Huang, C, Morgan, E, Gerstenfeld, L, **Schlezinger, J.** High fat diet leads to reduced fracture healing in a mouse model. *Orthopaedic Research Society Annual Meeting,* San Diego, CA, 2017.
19. Kim, S, Li, A, Monti, S, **Schlezinger, J.** Understanding tributyltin, an environmental obesogen, in its engagement of nuclear receptor pathways and molecular gene targets using transcriptomics. *Superfund Research Program Annual Meeting,* Durham, NC, 2016.
20. Edwards, L, Hatch, E, Janulewicz Lloyd, P, Webster, T, **Schlezinger, J**. Development of a PPARγ ligand exposure biomarker. *Superfund Research Program Annual Meeting*, Durham, NC, 2016.
21. Crawford, K, Nacci, D, Heiger-Bernays, W, S**chlezinger, J**. Using fish to simultaneously study the ecological and human health impacts of Superfund chemicals in New Bedford Harbor, Massachusetts. *Superfund Research Program Annual Meeting,* Durham, NC, 2016.
22. Watt, J, Baker, A, Meeks, B, Morgan, E, Gerstenfeld, L, **Schlezinger, J**. Tributyltin engages the RXR and LXR nuclear receptor pathways to modify osteoblast/osteoclast crosstalk and enhance bone deposition in a sex-dependent manner. *Society of Toxicology Annual Meeting*, New Orleans, LA, 2016.
23. Watt, J, Webster, T, **Schlezinger, J.** Strengths and limitations of Generalized Concentration Addition in modeling PPARγ activation by endocrine-disrupting compounds. *Society of Toxicology Annual Meeting*, New Orleans, LA, 2016.
24. Watt, J, Webster, T, **Schlezinger, J.** Predicting joint effects of PPARγ ligands using Generalized Concentration Addition. *Society of Toxicology Annual Meeting*, San Diego, CA, 2015.
25. Watt, J, Webster, TJ, **Schlezinger, J.** Predicting joint effects of PPARγ ligands using Generalized Concentration Addition. *Superfund Research Program Annual Meeting,* Baton Rouge, LA, 2014.

26. Watt, J, Webster, T, **Schlezinger, J.** Emerging toxicants induce adipogenesis and suppress osteogenesis in bone marrow multipotent mesenchymal stromal cells.

    Superfund Research Program Annual Meeting, Baton Rouge, LA, 2014.

    Society of Toxicology Annual Meeting, Phoenix, AZ, 2014.

27. Baker, AH, Watt, J, Meeks, B, Gerstenfeld, LC, **Schlezinger, JJ.** Environmental obesogens: are they bad to the bone? *Superfund Research Program Annual Meeting,* Raleigh, NC, 2012.

28. Bragdon, B, Morgan, EF, Burns, R, Baker, AH, Belkina, A, Denis, G, **Schlezinger JJ**, Gerstenfeld, LC. Age related sexual dimorphism of trabecular bone loss is inversely associated with adipogenic and osteoclastic but not osteogenic activities. *American Society of Bone and Mineral Research Annual Meeting*, Minneapolis, MN, 2012.

29. Bragdon, B, Burns, R, Baker, AH, Belkina, A, Dennis, G, Morgan, EF, Gerstenfeld, LC, **Schlezinger, JJ.** Role of Brd2 gene in the regulation of sex-linked bone loss and its association with adipocyte differentiation. *American Society of Bone and Mineral Research Annual Meeting*, Minneapolis, Minneapolis, MN, 2012.

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31. Baker, AH, Meeks, BD, Andrews, FV, Salazar, D, Gerstenfeld, LC, **Schlezinger, JJ.** Environmental contaminant tributyltin-induced osteoporosis: distinct involvement of PPARγ and RXR in suppression of osteogenesis. *American Society of Bone and Mineral Research Annual Meeting*, San Diego, CA, 2011.

32. Baker, A.H., Meeks, B.D., Mann, K.K., Gerstenfeld, L.C., **Schlezinger, JJ**. Dual PPAR/RXR agonist tributyltin uniquely impacts bone integrity and marrow microenvironment. *Society of Toxicology Annual Meeting*, Washington, DC, 2011.

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35. Yanik, SC, Sherr, DH, **Schlezinger, JJ.** Organotin-mediated PPARγ activation and adipocyte differentiation in bone marrow stromal cells. *Society of Toxicology Annual Meeting,* Salt Lake City, UT, 2010.

36. Haas, AR, Yanik, SC, Sherr, DH, Gerstenfeld, LC, **Schlezinger, JJ**. Tributyltin: B cell toxicant and bone marrow microenvironment modulator. *Society of Toxicology Annual Meeting,* Salt Lake City, UT, 2010.

37. **Schlezinger, JJ**, Haas, A, Sherr, DH. March Tributyltin: A dual bone marrow stromal cell and lymphocyte toxicant? *Society of Toxicology Annual Meeting,* Baltimore, MD, 2009.

38. Howard, GJ, **Schlezinger, JJ**, Webster, TF. A generalized concentration addition model predicts joint effects of TCDD and partial-agonist PCBs. *Society of Toxicology Annual Meeting*, Charlotte, NC, 2007.

39. **Schlezinger, JJ**, Bissonnette, S, Sherr, DH. GW7845, a PPARγ agonist, induces calcium-dependent MAP kinase activation and apoptosis in pro/pre-B cells. *Society of Toxicology Annual Meeting*, San Diego, CA, 2006.

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41. Liu, D, Emberley, J, Sherr, DH, **Schlezinger, JJ**. PPARγ agonists induce MAP kinase- but not calcium- or reactive oxygen species-dependent apoptosis in pro/pre-B cells. *Society of Toxicology Annual Meeting*, New Orleans, LA, 2005.
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47. **Schlezinger, JJ**, Blickarz, CE, Mann, KK, Stegeman, JJ. NF-κB (Rel) homologues in scup (*Stenotomus chrysops*) and possible activation of NF-κB by Ah receptor agonists. *International Symposium on Pollutant Responses in Marine Organisms*. Williamsburg, VA, 1999.
48. **Schlezinger, JJ**, Struntz, WDJ, Stegeman, JJ. Cytochrome P4501A (CYP1A) and planar halogenated aromatic hydrocarbons (pHAH): Oxidative inactivation and reactive oxygen production. *Society of Toxicology Annual Meeting*. Seattle, WA, 1998.
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ORIGINAL, PEER REVIEWED ARTICLES

1. **Schlezinger, JJ**, Bello, A, Mangano, KM, Biswas, K, Patel, PP, Pennoyer, EH, Wolever, TMS, Heiger-Bernays, WJ, Bello, D. 2025 Per- and poly-fluoroalkyl substances in circulation and their association with serum liver enzyme biomarkers in a Canadian population. *Environ. Health.* 24(1):10. PMCID: PMC11909942
2. Nielsen, G, Gondim, DD, Cave, MC, Heiger-Bernays, W, Webster, TF, **Schlezinger, JJ**. 2025. Perfluorooctanoic acid increases serum cholesterol in a PPARα-dependent manner in female mice *Archives Toxicol.* doi: 10.1007/s00204-025-03984-7. Online ahead of print. PMID: 40021516 2025 Mar 1.
3. **Schlezinger, JJ**, Biswas, K, Heiger-Bernays, WJ, Bello D. 2024. An oat fiber intervention for reducing PFAS body burden: a pilot study. *Toxicol. Appl. Pharm*. 495:117188. PMCID: PMC11798698

4. Sen, P, Fan, Y, **Schlezinger, JJ**, Hansen, T, Ehrlich, SD, Webster, TF, Hyötyläinen, T, Pedersen, O, Orešič, M. 2024. Exposure to environmental toxicants is associated with gut microbiome dysbiosis, insulin resistance and obesity**.** *Environ. Int.* 186:108569. PMID: 38522229

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ORIGINAL, ARTICLES ACCEPTED FOR PUBLICATION

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73. Ekuban, FA, Gripshover, TC, Ames, P, Bolatimi, OE, Wahlang, B, Abramson, J, Iyer, M, Luo, J, Ekuban, A, Watson, WE, Banerjee, M, Hwang, JY, Park, JW, Biswas, K, Bello, D, **Schlezinger, JJ**, McClain CJ, Cave, MC. Effects of Perfluorooctane sulfonate (PFOS) exposure on alcohol-associated liver disease severity in a chronic-binge ethanol feeding model. *Environ. Health Perspect*.

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MANUSCRIPTS IN PREPARATION

75. Nielsen, G, Reed, E, Webster, TF, Heiger-Bernays, W, Lara, B, Sherr, D, Hyötyläinen, T, **Schlezinger, JJ**. Integrated assessment of the effects of PFOA exposure on hepatic lipid profiles and transcriptomics in mice expression human PPARα. To be submitted to *Arch. Toxicol.*

76. **Schlezinger, JJ**. PPARα genotype modifies bone quality and the effect of perfluorooctanoic acid on bone quality. To be submitted to *Toxicol. Appl. Pharm.*

INVITED REVIEWS

1. **Schlezinger, JJ** and Gokce, N. 2024. Perfluoroalkyl/Polyfluoroalkyl Substances: Links to Cardiovascular Disease Risk. *Circ. Res.*. 134(9):1136-1159. PMCID: PMC11047059
2. Simmons AL, **Schlezinger JJ**, Corkey BE. 2014. What are we putting in our food that is making us fat? Food additives, contaminants, and other putative contributors to obesity? *Curr. Obesity. Rep.* 3(2): 273-285. PMCID: PMC4101898.

IARC MONOGRAPH

1. International Agency for Research on Cancer. *IARC monographs on the evaluation of carcinogenic risks to humans, volume 135. perfluorooctanoic acid and perfluorooctanesulfonic acid.* Lyon, France: IARC; 2024.

   I was the scientific expert that assessed more than 200 manuscripts related to human data (epidemiological and human in vitro models) on Receptor-Mediated Processes.

MANUSCRIPTS ON TEACHING PEDAGOGY

1. Furtunato E, Beard J, Bor J, Dolan CA, Halim N, Onyango MA, Peters JL, **Schlezinger J**, Godley S. Balancing Autonomy and Equity in Core Courses: Recommendations for Teaching Teams and Administrators of Schools and Programs of Public Health. Accepted for publication in *Pedagogy Heal. Promot.*

PATENTS

USPTO #61368042: Aryl hydrocarbon receptor modifiers as novel cancer therapeutics. Patents granted in Australia, Japan, China; pending in U.S., Brazil, Canada, Eurasia, Europe, India.

PUBLICALLY AVAILABLE DATASETS

1. GSE119541. Analyses published in Kim et al., 2018.

RESEARCH TRANSLATION

1. On petition for review from final rule of the United States Environmental Protection Agency 89 FED. REG. 32, 532 (Apr. 26, 2024). Brief for Dr. Linda Birnbaum, PH.D., Dr. Jamie Dewitt, PH.D., Dr. Rainer Lohmann, PH.D. and Dr. Jennifer Schlezinger, PH.D, as AMICI CURIAE in Support of Respondents. January 2025.
2. Interview with WBRU on per- and polyfluoroalkyl substances (PFAS) and their toxicity. January 2023.
3. Review of California Public Health Goals for PFOA/PFOS in Drinking Water. February 2022.
4. Submitted comments on the documents prepared by the US Environmental Protection Agency for review by the Science Advisory Board: Per- and polyfluoroalkyl substances (PFAS) Review Panel Environmental Protection Agency. December 2021.
5. Submitted comments in support of the proposed amendments to 301 CMR 41: Toxic or Hazardous Substance List (TURA List) including adding Per- and Polyfluoroalkyl Substances Not Otherwise Listed (PFAS NOL) to the Toxic or Hazardous Substance List and a definition of the term "substance" to the regulation as a means of clarification. October 2021.
6. Invited webinar for the Northeast Waste Management Official's Association. *PFOA induces serum and liver dyslipidemia*. March 2021.
7. Submitted comments in support of the Massachusetts Department of Environmental Protection's (MassDEP) derivation of the Maximum Contaminant Level (MCL) for PFAS. February 2020.
8. MA Department of Environmental Protection presentation titled "Adverse Effects of Environmental Contaminants on Metabolic Health. What may new flame retardants and PFAS have in common?" June 2018.
9. Contributor to: Miller M, Schettler T, Tencza B, Valenti M. *A Story of Health*. *Infertility.* Agency for Toxic Substances and Disease Registry, Collaborative on Health and the Environment, Science and Environmental Health Network, Western States PESHU. 2017.
10. Superhuman Radio interview (*What are we putting in our food that is making us fat?*)
11. Museum of Science podcast (*Chemicals, Osteoporosis and Obesity*)
12. Collaborative on Health and the Environment partnership calls
    - *25 Years of the Superfund Research Program: Highlights and Hopes*
    - *Home Invaders: Are flame retardants fattening us up and harming our bones?*
    - *Fatty Bones Make Bad Skeletons: Influence of Bone-disrupting Chemicals across the Lifespan*